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INSECTS, BOTH HOSTS AND VECTORS OF PLANT VIRUSES[1]

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Man, in his attempts to explain the observed phenomena of nature, travels from naive conjectures to the complexities of experimental facts and then seeks generalized simplicity. In the field of virus research we are still in the complex stage in which every generalization proposed has at least one Achilles Heel. A Newton of viruses has not yet appeared on the horizon. Nevertheless, the chase is on and in scores of laboratories throughout the world the quarry is being glimpsed at closer and closer, though still distant, range. Most avidly pursued of all is tobacco mosaic virus which must scarcely dare call its nucleic acid its own.

Among the most complex and least understood of the plant viruses are those which rely heavily or exclusively on insects for their dissemination in nature. The relationships of viruses to their vectors have been studied by a relatively small group of research workers. Yet evidence is accumulating that the insect vector, far from being merely a "flying needle" contaminated with virus, as some have been inclined to consider it, may prove to be the pristine host of some, if not all, plant viruses.

I should like to review some of the more interesting discoveries and hypotheses involving vector-virus relationships, particularly the recent ones and those dealing primarily with the leafhopper transmitted viruses.

Some important discoveries have been made accidentally or as by-products of planned investigations; others have required the penetrating thought and imagination so perceptively described by Schopenhauer (and recently quoted by a colleague) when he said: "Thus the task is, not so much to see what no man has seen yet, but to think what nobody has thought yet, about that which everybody sees."

Why is a Vector a Vector?

The vast majority of known plant virus vectors are insects. Eriophyid mites are also proving to be important vectors of a

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few viruses in recent years. This year for the first time, a non-arthropod was demonstrated to transmit a plant virus when Hewitt *et al.* (1958) reported *Xiphinema index* Thorne and Allen, a plant parasitic nematode, to be a vector of grape fan leaf virus.

One of the basic questions in vector-virus relationships, and one about which we are still almost completely ignorant, concerns the factors which govern the transmissibility of viruses by vectors. The vectors of most plant viruses show general but not inflexible phylogenetic affinities. Thus, aphid-transmitted viruses have not been shown conclusively to be transmitted also by leafhoppers, beetles or other insect groups. Viruses associated with leafhoppers are transmissible only by leafhoppers—except in the case of Pierce's disease virus of grapes which is carried by spittle bugs of the family Cercopidae and by sharpshooters in the family Cicadellidae, but not by other leafhoppers. The common denominator among all the vectors of this virus is their habit of feeding in the xylem rather than the phloem of the plants.

There appears to be less group specificity among the vectors of animal viruses. For example, the experimental vectors of the virus causing western equine encephalitis include ticks as well as mosquitoes.

Bennett and Wallace (1938) have shown that the curly top virus of sugar beets, transmitted in the United States only by *Circulifer tenellus* (Baker), can be acquired and carried in the bodies of aphids, mites, thrips and other leafhoppers for long periods of time, but is not transmitted by these species.

The specific cause for failure of a virus to be transmitted by non-vector species or by a genetic strain of its normal vector has been demonstrated only once. Storey (1939) bred a strain of *Cicadulina mbila* (Naudé) which consistently failed to transmit maize streak virus in Africa. However, these "inactive" individuals could be made active vectors if, after they had fed on a diseased plant, the stomach wall of the insects was punctured with a fine needle to permit some of the infective plant juice to enter the blood in the body cavity. This showed that the barrier to virus passage existed in the wall of the intestine.

Present evidence suggests that the constitutional stability of insects as vectors of plant viruses is much greater than are the viruses which they transmit. However, this does not imply that

vector species are homozygous in this respect and show uniformity and consistency in transmitting ability.

Björling and Ossiannilsson (1958) have just published the results of extensive experiments involving 10 asexually reared strains of six different species of aphids as vectors of two persistent viruses, beet yellows and potato leaf roll. The aphids were: *Myzus persicae* (Sulzer), *M. ascalonicus* Doncaster, *Aphis fabae* Scopoli, *Aulacorthum circumflexum* (Buckton), *A. solani* Kaltenbach) and *Macrosiphum euphorbiae* (Thomas). Nearly all of the strains of *Myzus persicae* were better vectors of both potato leaf roll and beet yellows virus than were the best strains of the other aphid species compared.

Most of the work dealt with 85 strains of *Myzus persicae* which had been collected from widely different geographical and ecological sources. Virus transmission tests over a three year period revealed that these 85 strains could be grouped in a continuous series which ranged from 10 per cent to 80 per cent in virus transmitting ability. Moreover, there was no correlation between the field source plant of the aphid strain, nor the plants on which the aphids fed just prior to being used in virus tests, and their efficiency as vectors.

Most of the strains were fairly efficient vectors with individual aphids of 70 of the 85 strains transmitting beet yellows virus to 30 per cent or higher of the test plants. The absolute differences in transmission efficiency were great between some strains and significant in a number of cases. The authors were led to conclude that "these differences between more or less effective vector strains within the same species seem to be genetically determined." This was also supported by tests in which two aphid strains of similar vector efficiency were crossed. The efficiency of the progeny was markedly below that of the parents.

Loss and Recovery of Insect Transmissibility by Viruses

There is now considerable evidence that the insect transmissibility of many plant viruses is not constant, but varies sometimes in relatively short periods of time. For many years it has been the not uncommon experience of those who work with insects and viruses to find that known vectors of certain viruses sometimes transmit them inefficiently or not at all. Very often this is encountered after an interruption of transmission work for several months or years. It is apparently often due to a reduction in virus

titre in a plant which has been infected for an extended period of time. However, in other instances it may occur with viruses whose titre has been maintained at a high level by frequent transmission to new plants or animals by means of mechanical inoculation.

Transmissibility by mosquitoes of certain strains of yellow fever, encephalitides and dengue viruses is greatly reduced or eliminated after repeated passage by mechanical means in the vertebrate hosts (Reeves, 1958).

Viruses are known to mutate and it is probable that vectorless mutants may appear which, in the absence of insect transmission, might supplant the insect transmissible virus strain.

Black (1953) maintained three isolates of potato yellow dwarf virus for 12-16½ years in plants without insect transmission. At the end of this period the leafhopper vector was unable to transmit these isolates whereas control leafhoppers transmitted fresh isolates of the virus, collected in the field, with normal high frequency. Very recently Black *et al* (1958) reported the same phenomenon for wound-tumor virus and its leafhopper vector.

Similar loss of transmissibility by aphids was reported by Hollings (1955) for tomato aspermy virus. One isolate, at first easily transmitted by aphids, was transmitted only with great difficulty by vectors after two years of mechanical transmission to tobacco.

I worked with an aphid transmissible virus of garden nasturtium, *Tropaeolum majus,* over a period of several years. Vector work was then discontinued for approximately two years during which the virus was transmitted by juice inoculation every few months. When aphids were tried again as vectors transmission occurred only rarely. Field collected *Myzus persicae* were also tried as vectors but they succeeded no better than did our greenhouse colonies.

In the above cases loss of transmissibility by insects followed two or more years of virus maintenance or transmission in the absence of insects. Swenson (1957) reported that one of two isolates of bean yellow mosaic virus lost its ability to be transmitted by three species of aphids within a period of one to four months.

The most obvious explanation suggested by these results is that the virus isolates in question lost their ability to be trans-

mitted by their normal vectors as a result of mutations of the virus. Since the tomato aspermy virus isolate became almost completely vectorless in a two-year period it may be inferred that the alleged mutant strain was replacing the insect transmitted strain gradually. In contrast to this, however, is the fact that Swenson's bean virus apparently became completely vectorless within one to four months after having been transmitted readily by three species of aphids.

That the apparent loss of insect transmissibility by a virus may be reversible has been reported by Watson (1956, 1958). Potato virus C, derived from potatoes, was not transmissible by aphids. However, after mechanical inoculation into *Nicotiana glutinosa* this virus could be transmitted by *Myzus persicae*. When returned to potato by means of aphids, the virus ultimately reverted to the vectorless strain although certain isolates remained aphid transmissible through several passages in potato.

The first speculative explanations suggested by this phenomenon were of two different types. One involved possible mutations; the other required a quantitative rather than a qualitative hypothesis and postulated vectorial and vectorless virus particles which varied in ratio in the different hosts. Each of these hypotheses required assumptions that are without precedent.

What may prove to be a more plausible explanation has just been reported. Badami and Kassanis (1958) obtained a strain of potato virus Y, from *Solanum jasminoides* from India, which in potato closely resembled the potato virus C used by Watson. However, after separation from two other viruses which were also present in *S. jasminoides*, this virus was readily transmitted by *Myzus persicae*. These previously undescribed viruses are reported to decrease the multiplication of the virus Y strain and also its aphid transmissibility.

This suggests that Watson's potato virus C may not be aphid-transmissable from potato because of the possible presence in potato of another virus which reduces or prevents transmission of virus by aphids. Tobacco may not be a host of this virus and thus virus C would be available for transmission by aphids.

This phenomenon is just the opposite of that reported by Smith (1945) for the virus complex known as tobacco rosette. One component, mottle virus, is easily transmitted mechanically but

cannot be transmitted by *Myzus persicae* unless it occurs in the same plant which carries vein-distorting virus.

Cross Protection Between Viruses in Insects

Although plants are not known to produce antibodies to infective disease agents such as we know to be common in animals, plants, after infection by a virus, may acquire immunity to related and sometimes more virulent strains of the same virus. The different strains have been assumed to arise through mutation, but there is now evidence that genetic recombination may also be involved. The ability of one virus strain to prevent a second strain from producing its own characteristic disease symptoms in the same plant has been considered evidence that the two strains are closely related. However, it is also possible to have closely related strains, such as those of curly top virus of sugar beets, which do not protect against each other either in the plant or in the insect vector (Giddings, 1950).

Kunkel (1955) showed that the California and the eastern U.S. strains of aster yellows virus protect against each other in some of their common host plants. He also obtained evidence that the same strains protect against each other in the aster leafhopper, *Macrosteles fascifrons* (Stål). Working with groups of leafhoppers, he found that those which fed for two weeks on plants infected with one strain of virus and then fed two weeks on plants carrying the other virus strain, always transmitted only the first strain.

Experiments of several different types have demonstrated that aster yellows virus multiplies in the aster leafhopper as well as in the plant. Although the mechanism of cross protection among viruses is not known, it has been inferred that related strains need the same materials for multiplication and that the first virus to invade a host occupies the sites of virus multiplication and uses up the materials available, thus denying to the challenging strain the products it needs for multiplication.

However, more extensive experiments by Freitag (1958) in California with three strains of aster yellows virus and the aster leafhopper have shown that cross protection is not always stable and permanent. Working with single insects and transferring them daily over long periods of time, he found that under some circumstances the leafhoppers finally stopped transmitting the first strain acquired and then transmitted the challenging strain. These were,

however, exceptional cases. In most instances there was marked cross protection in both insect and plant.

Maramorosch (1957, 1958a) has reported the same phenomenon with two strains of corn stunt virus and its vector *Dalbulus maidis* (DeLong & Wolcott). In the studies conducted with aster yellows virus and with corn stunt virus, one strain in each disease complex showed greater dominance than the other in both the insect vector and the host plant. In the leafhoppers, strain "A", acquired first, was sometimes replaced by strain "B". However, if strain "B" was acquired first, strain "A" failed to supplant it. This indicates that the phenomenon of cross protection is more complex than mere prior occupancy of virus multiplication centers in the insect or plant by the first strain to enter.

Our perplexity regarding the nature of this apparent competition between plant virus strains within the body of the leafhopper vector is compounded by the nature of strain replacement in the insect. In a few instances, test plants fed on by single insects showed symptoms of both virus strains. In most cases, however, symptoms were entirely of one strain or the other. Also, replacement of one strain by a more dominant strain within the same leafhopper was not always permanent when first accomplished. Sometimes the strains were transmitted alternately before the dominant strain finally completely supplanted the other.

The presence and multiplication of two virus strains in the same leafhopper vector have not yet been known to result in a new hybrid virus. However, more extensive tests involving related virus strains carried by the same vectors might produce new hybrid plant viruses in insects as it has in plants and in vertebrate animals.

Recombinations of genetic units between related strains of animal and plant viruses have already been reported. The first work of this type was done by Burnet and Lind (1951) with strains of influenza virus. Subsequently, Best and Gallus (1955) and Best (1956) achieved similar results with tomato spotted wilt virus. Plants infected with mixtures of two strains of this virus developed symptoms of both parent strains and also symptoms of new strains. From these plants with mixed infections they recovered both parent strains and also several new strains. The latter were distinct from each of the parent strains but possessed some of the characters of each parent. Further experiments led

to the conclusion that such new strains were genetically stable and not mere mixtures of the two parent strains.

It would be of great interest to determine if parallel results could be obtained by allowing the thrips vector to acquire two or more of these same strains of spotted wilt virus. Multiplication of spotted wilt virus in thrips has not yet been demonstrated, but the 5–10 day incubation period of the virus in the vector, plus long retention of the virus by the vector, suggest multiplication.

Beneficial Effects of Plant Virus in Relation to Insect Vectors

A few reports exist indicating that a plant virus may be of indirect benefit to its own insect vector or to a non-vector species. Carter (1939) found that infected *Emilia sonchifolia*, a weed host of both spotted wilt virus and its vector *Thrips tabaci* Lindeman, maintained a higher population of thrips than did healthy plants. The reasons for this appeared to be that diseased plants had curled leaves which provided improved shelter for the thrips vector and that these plants also grew more slowly and survived longer.

A more direct beneficial effect of virus on *Aphis fabae* has been reported by Kennedy (1951). He found that sugar beet mosaic virus altered the physiology of the beet plant in such a manner that the production of young aphids per mother averaged higher on virus infected leaves of all ages than on healthy leaves of comparable age.

Severin (1946), incidental to his search for new leafhopper vectors of aster yellows virus, found that 9 species of leafhoppers completed their nymphal development on celery or asters infected with California aster yellows virus, but that the adults died within a few days after transfer to healthy celery or asters. No data were presented on the longevity of any control adults that may have been held on diseased plants. The results given were interpreted as demonstrating that aster yellows virus in some way altered celery and aster plants so as to make them more suitable food hosts for the leafhopper species tested.

Maramorosch (1958b) reported similar conclusions drawn from experiments with *Dalbulus maidis* (DeLong & Wolcott), a non-vector of aster yellows virus. This leafhopper, which transmits corn stunt virus, was known to feed well and breed only on *Zea mays* and *Euchlaena mexicana*. Eighty adult insects were caged on healthy China asters and 80 were caged on asters infected with

aster yellows virus. All insects on healthy asters were dead within four days, but 63 of 80 were still alive on virus-infected plants after 42 days. The survival on diseased asters was practically identical with the survival on corn plants but no eggs were deposited in the aster plants. In subsequent tests *D. maidis*, after five weeks on infected asters, were successfully maintained on healthy asters. Adults and nymphs of a related leafhopper, *D. elimatus* (Ball), with a limited host range identical to that of *D. maidis*, were found to survive well on asters carrying virus, but died rapidly on healthy asters. Maramorosch interprets his experiments as indicating that the virus altered the chemical composition of the aster plant in the direction of more adequate nutrition for the leafhoppers which, in the case of *D. maidis*, became sufficiently adapted to aster as to permit survival even on virus-free asters.

Among the species discussed by Severin (1946) in his report on the longevity of leafhoppers on virus-infected and healthy plants was *Texananus spatulatus* Van Duzee. He stated that individuals of this species which transmitted aster yellows virus lived longer than those which failed to transmit virus. Only a small part of the data on which this conclusion was based has been published (Severin, 1945). Since the virus transmitting efficiency of this species was exceedingly low (most individuals carrying virus infected less than one percent of the plants fed upon) and since it is not clear whether the transmitters and the non-transmitters were maintained in the same manner, further confirmation is needed for the generalization that the transmitting insects lived longer than the non-vectors. Severin construed the increased longevity to mean that the plants infected were better hosts and prolonged the life of the leafhoppers. However, since the insects were transferred daily to new test plants and transmitted virus to only a few plants this explanation does not seem feasible. If the transmitting insects lived significantly longer than non-transmitters, it would be evidence that the virus *per se* was beneficial to the insect.

No reports, parallel to those given for plant viruses, have been found to indicate that animal viruses may render their hosts suitable as food sources for arthropods if, in the absence of virus, such hosts are unacceptable to the arthropod species. Virus-infected animals sometimes carry abnormally high populations of arthropod parasites but apparently this is only because such animals become

debilitated and therefore are less capable of ridding themselves of the parasites than are healthy individuals.

In none of the situations just described was it suggested by the research workers involved that the virus itself contributed directly to the increased longevity of the insect vector.

A Virus Harmful to Both Animal and Plant

Although there have been many insect-borne plant viruses known and a less lengthy list of insect-borne animal viruses, until recently there has been no report of a virus that was harmful to its insect vector.

It is true that we have such an example among the rickettsiae, some of which have also been called viruses, but Snyder (1948) classes these as "micro-organisms . . . intermediate in characteristic between bacteria and viruses . . ." The body louse, vector of typhus fever rickettsiae, is also killed by the infectious agent it carries. Zinsser (1935), describes the plight of the body louse in these words: "The louse shares with us the misfortune of being prey to the typhus virus. If lice can dread, the nightmare of their lives is the fear of some day inhabiting an infected rat or human being. For the host may survive; but the ill-starred louse that sticks his haustellum through an infected skin, and imbides the loathsome virus with his nourishment, is doomed beyond succor. In eight days he sickens, in ten days he is *in extremis*, on the eleventh or twelfth his tiny body turns red with blood extravasated from his bowel, and he gives up his little ghost."

The first indication that a plant virus could cause tissue changes in its insect vector was given by Littau and Maramorosch (1956). They reported that aster yellows virus caused a higher percentage of fat body cells to have stellate nuclei than occurred in virus-free *Macrosteles fascifrons* (Stål). There is as yet, however, no evidence that these effects are harmful to the leafhopper.

The effect of aster yellows virus on the longevity of this vector was tested extensively by Severin (1947). Both infective and non-infective leafhoppers were held on Sacramento barley, which is immune to the virus, and survival was recorded. Adult longevity ranged from approximately 30 days to over 140 days. The 350 infective leafhoppers survived as long as did an equal number of non-viruliferous individuals. He concluded that the virus itself is neither beneficial nor injurious to adult aster leafhoppers.

A report from Japan (Yoshii and Kiso, 1957), indicates that

in several respects the virus causing dwarf disease of orange alters the metabolism of infected orange leaves and the metabolism of the plant hopper vector, *Geisha distinctissima* Wal., in a similar manner. For example, oxygen consumption and total phosphorus were reduced in both host plant and vector. The publication does not indicate whether or not these metabolic disturbances adversely affected the infective insects in a way that was reflected in reduced longevity, reproduction or activity.

Recently we have been able to demonstrate that the most important stone fruit virus in western North America, Western X-disease virus, causes the premature death of at least one of its leafhopper vectors.

Colladonus montanus (Van Duzee) had proved to be an exceedingly inefficient vector of the virus from peach to peach. However, after the discovery that celery is also a susceptible plant host of the virus (Jensen, 1956), *C. montanus* was retested using celery as the virus source. From these experiments it was determined that *C. montanus* readily acquires the virus from celery. Groups of leafhoppers, fed alternately on a series of healthy peach and celery plants, transmitted virus to 57 of 213 celery plants but to only four of 238 peach trees (Jensen, 1957). Because of the superiority of celery over peach as both a virus source and a test plant and because *C. montanus* lives well on celery but dies rapidly on peach, experiments comparing the longevity of viruliferous and non-viruliferous leafhoppers were carried out on celery. Longevity was measured from the first day on test plants which was after completion of the average latent period of the virus in the vector.

The experiments, reported elsewhere (Jensen, 1958; 1959), show that Western X-disease virus itself causes the premature death of its insect vector. The leafhoppers which did not transmit virus survived on the test plants approximately twice as long as did the transmitters. The increased mortality was not due to altered plant nutrition, because in many of the tests all insects, both transmitters and non-transmitters, fed together on the same virus source plants. The single variable was whether or not a given individual happened to acquire virus from the diseased plant upon which all had fed.

Moreover, an alternative explanation cannot be found in inherent differences between individual leafhoppers. The percentage of insects proving to be infective was usually directly proportional to the access time on the inoculum plant. Thus, in one experiment

insects from the same stock colony were caged on a diseased plant and removed from the plant as three different groups each with a different acquisition feeding time. Of those removed after three days, 27 per cent transmitted virus. After seven days, 70 per cent transmitted and after 16 days 90 per cent transmitted virus. In each group, longevity was greatest among the non-transmitters.

Multiplication of Virus in Insect Vectors

Multiplication of persistent viruses in their aphid vectors has not yet been demonstrated conclusively. Day (1955) reported experiments which suggested to him that potato leaf roll virus multiplies in the aphid *Myzus persicae*. However, Harrison (1958) found that infectivity and retention of this virus by *M. persicae* increased with feeding time on a virus source and that aphids gradually lost their virus on immune hosts. He concluded that the virus does not multiply in the aphid.

It has now been well-established that four plant viruses do multiply in their leafhopper vectors as well as in their plant hosts. These are the viruses of rice stunt, clover club-leaf, aster yellows and wound tumor.

Fukushi (1939) demonstrated that rice stunt virus is transmitted transovarially by its vector *Nephotettix apicalis* Motschulsky var. *cincticeps* Uhler. Starting with a single infective female, the experiment was carried through six leafhopper generations over a period of 374 days. At the end of the experiment there was no evidence of a decline in the number of insects per generation becoming infective nor in the percentage of plants infected.

Black (1950) carried out an experiment, similar to that of Fukushi, with clover club-leaf virus and its vector *Agalliopsis novella* (Say). Starting with a single viruliferous female, Black maintained the insects for five years and through 21 generations on alfalfa which is immune to the virus. At the end of this period there was no reduction in virus infectivity in the insects. If there was no multiplication in the insects the virus would have to retain its infectivity at a dilution conservatively estimated to exceed $1:2.8\times10^{26}$. This is far beyond the dilution tolerance of the virus and means that the virus multiplies in the vector.

Occasional transmission of plant virus through the egg of the insect vector has also been reported by Black (1953) for wound-tumor virus and potato yellow dwarf virus in their respective vectors, *Agalliopsis novella* and *Agallia constricta* Van Duzee. Also,

Grylls (1954) reported that rugose leaf curl virus is transmitted through the egg of its leafhopper vector, *Austroagallia torrida* Evans. It is not yet known whether these viruses can persist in their vectors generation after generation, as has been demonstrated for the viruses causing rice stunt and clover club leaf, without being replenished by feeding on a diseased plant.

That aster yellows virus (Black, 1941; Maramorosch, 1952) and wound tumor virus (Black & Brakke, 1952) multiply in their respective leafhopper vectors has been shown by serial passage of the viruses through their vectors. Diluted virus was injected mechanically into the bodies of the vectors. Later, virus was recovered from the same insects, diluted, and injected into fresh leafhoppers. Ten such serial passages were made with aster yellows virus and seven with wound tumor virus, yet the concentration of virus in the insects attained as high a level in the last passages as in the first. In the absence of multiplication one would have to postulate a dilutions of 10^{-40} for aster yellows virus and 10^{-18} for wound tumor virus.

It is probable that among the many other leafhopper transmitted viruses, especially those having long incubation periods in the vector, will be found additional plant viruses which also multiply in insects. However, it should not be assumed that all leafhopper vectors are also virus hosts. Freitag (1936) and Bennett and Wallace (1938) have presented strong evidence that curly top virus of sugar beets does not multiply in the beet leafhopper, *Circulifer tenellus* (Baker). It should be added, however, that this virus has a very short (approximately 24 hours) incubation period in the vector.

Virus Origin

After tobacco mosaic virus was purified and shown to be a crystalline protein (Stanley, 1935), viruses were considered by some to be unrelated to living organisms. It was suggested that plant viruses originated first from plant cell components and secondarily developed the broad spectrum of specialization which ranges from little or no dependence upon insect vectors, such as in the case with tobacco mosaic virus, up to the leafhopper transmitted viruses, most of which require insects for their dissemination.

In opposition to this idea, is the organismal theory of virus origin proposed by Green (1935) and supported by Laidlaw

(1938), Andrewes (1952) and others. According to this view viruses originated by retrograde evolution from parasitic microorganisms possibly akin to protozoa and bacteria. The fact that we now have several viruses which multiply in animals (insects) as well as in plants has brought into greater prominence the organismal theory of virus origin, and has resulted in its extension (Maramorosch, 1954) to suggest that plant viruses were originally arthropod viruses.

Viruses had been observed to cause disease symptoms in the plant hosts, but not in the insect vectors. Until plant viruses were demonstrated to multiply in the insect vector, their association with insects had been considered somewhat fortuitous. However, with the knowledge that some insects are hosts and not merely vectors of these viruses the way was open to new interpretations as to virus origin.

Among the widely held generalizations in biology is one stating that those parasite relationships which are characterized by severe damage to the host are of relatively recent origin whereas those in which the parasite lives at the expense of the host but causes the latter little or no damage are of much greater antiquity. Thus plant hosts, such as those of aster yellows virus, which suffer extreme damage, would be considered recently acquired hosts. The leafhoppers in which this virus also multiplies without apparent harm would be considered primitive hosts. Andrewes (1957) has reviewed this subject extensively and suggests that arthropods may have been the original hosts not only of the insect viruses, but also of viruses commonly associated with plants and with vertebrates. Insects play a critical role in all three of these general groups of viruses. Originally viruses may have had insects as their only hosts. Later the viruses may have made use of vertebrates and plants to get from one insect to another. Lest we become too carried away by this argument, however, it should be pointed out that in the host range of some plant viruses and some vertebrate viruses there are species which are as symptomless as are the vectors which transmit the viruses. Nonetheless, this does not invalidate the theory. It merely means that such viruses may have been associated with the symptomless hosts much longer than with those still suffering damage. Andrewes (1957) points out that epidemics of yellow fever disease occur in South American monkeys but not in African monkeys. The virus appears to be

harmless to African monkeys and for this reason it may be inferred that the virus has existed in Africa much longer than in the New World.

Similar examples occur among plant viruses. The virus causing Pierce's disease in grapes is probably of New World origin because its effect on the native American species of *Vitis* is mild whereas it is lethal to the introduced *Vitis vinifera* Linnaeus, the grape of history, which is considered to be native from southeastern Europe to western India. Also in America is a long list of apparently symptomless host plants of Pierce's disease virus, ranging from grasses to woody shrubs. Moreover, in America are many species and high populations of "sharpshooters," the leafhopper vectors of Pierce's disease virus. This group of leafhoppers is almost without representation in Europe.

The recent discovery of a plant virus which causes harm to its insect vector adds support to the theory of an insect origin of plant viruses. It provides more conclusive evidence that certain plant viruses are also animal viruses. Moreover, it supplies a concrete example of one stage in the postulated evolution of a virus which until now existed only in theory. It may even raise a doubt, however microscopic, regarding the validity of one of our oldest generalizations in virology, namely, that humans and other vertebrates are immune to injury by plant viruses.

Literature Cited

Andrewes, C. H.

1952. The place of viruses in nature. Proc. Roy. Soc. B139:313–326.

1957. Factors in virus evolution. Advances in Virus Research 4:1–24.

Badami, R. S. and B. Kassanis

1958. In: Report of the Rothamsted Experimental Station for 1957. (fide: Rev. Appl. Mycology 37:631.)

Bennett, C. W. and H. E. Wallace

1938. Relation of the curly top virus to the vector, *Eutettix tenellus*. Jour. Agr. Res. 56:31–52.

Best, R. J.

1956. Living molecule: A survey of recent advances in our understanding of the nature of viruses. Austral. Jour. Sci. 18 (4A):106–120.

Best, R. J. and H. P. C. Gallus

1955. Further evidence for the transfer of character-determinants (recombination) between strains of tomato spotted wilt virus. Enzymologia 17:207–221.

Björling, K. and F. Ossiannilsson

1958. Investigations on individual variations in the virus-transmitting ability of different aphid species. Socker. Handlingar II. 14(1): 1–13.

BLACK, L. M.
1941. Further evidence for multiplication of the aster-yellows virus in the aster leafhopper. Phytopathology 31:120–135.
1950. A plant virus that multiplies in its insect vector. Nature 166: 852–853.
1953. Loss of vector transmissibility by viruses normally insect transmitted. Phytopath. 43:466.

BLACK, L. M. AND M. K. BRAKKE
1952. Multiplication of wound-tumor virus in an insect vector. Phytopathology 42:269–273.

BLACK, L. M., SYLVIA WOLCYRZ AND R. F. WHITCOMB
1958. A vectorless strain of wound-tumor virus. VIIth Internat. Congress for Microbiology, Abstracts p. 255.

BURNET, F. M. AND P. E. LIND
1951. A genetic approach to variation in influenza viruses. 4. Recombination of characters between the influenza virus A strain NWS and strains of different serological subtypes. Jour. gen. Microbiol. 5:67–82.

CARTER, W.
1939. Populations of *Thrips tabaci* Lind. with special reference to virus transmission. Jour. Animal Ecology 8:261–276.

DAY, M. F.
1955. The mechanism of the transmission of potato leaf roll virus by aphids. Austral. Jour. Biol. Sci. 8(4):498–513.

FREITAG, J. H.
1936. Negative evidence on multiplication of curly-top virus in the beet leafhopper, *Eutettix tenellus*. Hilgardia 10(9):303–342.
1958. Cross-protection tests with three strains of the aster yellows virus in host plants and in the aster leafhopper. Phytopath. 48(8):393.

FUKUSHI, T.
1939. Retention of virus by its insect vectors through several generations. Proc. Imp. Acad. (Japan). 15(5):142–145.

GIDDINGS, N. J.
1950. Some interrelationships of virus strains in sugar-beet curly top. Phytopathology 40(4):377–388.

GREEN, R. G.
1935. On the nature of filterable viruses. Science 82:443–445.

GRYLLS, N. E.
1954. Rugose leaf curl—A new virus disease transovarially transmitted by the leafhopper *Austroagallia torrida*. Austral. Jour. Biol. Sci. 7:47–58.

HARRISON, B. D.
1958. Studies on the behavior of potato leaf roll and other viruses in the body of their aphid vector *Myzus persicae* (Sulz.). Virology 6(1):265–277.

HEWITT, W. B., D. J. RASKI AND A. C. GOHEEN
1958. Nematode vector of soil-borne fanleaf virus of grapevines. Phytopathology 48(11):586–595.

HOLLINGS, M.
1955. Investigation of Chrysanthemum viruses. I. Aspermy flower distortion. Ann. Appl. Biol. 43(1):86–102.

JENSEN, D. D.
1956. Insect transmission of virus between tree and herbaceous plants. Virology 2:249–260.
1957. Differential transmission of peach yellow leaf roll virus to peach and celery by the leafhopper, *Colladonus montanus*. Phytopath. 47(10):575–578.
1958. Reduction in longevity of leafhoppers carrying peach yellow leaf roll virus. Phytopath. 48:394.
1959. A plant virus lethal to its insect vector. Virology (in press).

KENNEDY, J. S.
1951. Benefits to aphids from feeding on galled and virus-infected leaves. Nature 168:825.

KUNKEL, L. O.
1955. Cross protection between strains of yellows-type viruses. Advances in Virus Research 3:251–273.

LAIDLAW, P. P.
1938. Virus diseases and viruses. Cambridge Univ. Press. Cambridge, England.

LITTAU, V. C. AND K. MARAMOROSCH
1956. Cytological effects of aster-yellows virus on its insect vector. Virology 2:128–130.

MARAMOROSCH, K.
1952. Direct evidence for the multiplication of aster-yellows virus in its insect vector. Phytopathology 42:59–64.
1954. Biological transmission of plant viruses by animal vectors. Trans. N. Y. Acad. Sci. Ser. II, 16:189–195.
1957. Cross-protection studies of two types of corn stunt virus. Phytopath. 47:23.
1958a. Cross protection between two strains of corn stunt virus in an insect vector. Virology 6:448–459.
1958b. Healthy and yellows asters as food plants for corn leafhoppers. VIIth Internat. Congress Microbiol.: 260.

REEVES, W. C.
1958. Arthropods as vectors and reservoirs of animal pathogenic viruses. In: Handbuch der Virusforschung 4:177–202. Springer-Verlag. Wien.

SEVERIN, H. H. P.
1945. Evidence of nonspecific transmission of California aster-yellows virus by leafhoppers. Hilgardia 17(1):21–59.
1946. Longevity, or life histories, of leafhopper species on virus-infected and on healthy plants. Hilgardia 17(3):121–137.
1947. Longevity of noninfective and infective leafhoppers on a plant nonsusceptible to a virus. Hilgardia 17(16):541–543.

SMITH, K. M.
1945. Transmission by insects of a plant virus complex. Nature 155:174.

SNYDER, J. C.
1948. The typhus fevers. In: Viral and Rickettsial Infections of Man. Edited by T. M. Rivers. J. B. Lippincott Co., Philadelphia.
STANLEY, W. M.
1935. Isolation of a crystalline protein possessing the properties of tobacco mosaic virus. Science 81:644–645.
STOREY, H. H.
1939. Transmission of plant viruses by insects. Bot. Rev. 5: 240–272.
SWENSON, K. G.
1957. Transmission of bean yellow mosaic virus by aphids. Jour. Econ. Ent. 50(6):727–731.
WATSON, MARION A.
1956. The effect of different host plants of potato virus C in determining its transmission by aphids. Ann. Appl. Biol. 44 (4):599–607.
1958. The specificity of transmission of some non-persistent viruses. Proc. 10th Internat. Congress of Ent. Montreal, August, 1956, 3:215–219.
YOSHII, HIROMU AND AKIRA KISO
1957. Studies on the nature of insect-transmission in plant viruses (II). Some researches on the unhealthy metabolism in the viruliferous plant hopper, *Geisha distintissima* Wal., which is the insect vector of the dwarf disease of Satsuma orange. Virus 7(5):315–320.
ZINSSER, H.
1935. Rats, Lice, and History. Boston, Little, Brown and Co.

PYRAMIDOBELA ANGELARUM KEIFER ON ORNAMENTAL BUDDLEIA IN THE SAN FRANCISCO BAY AREA

(Lepidoptera: Ethmiidae)

Pyramidobela angelarum was described in 1936[1] from southern California. Specimens had been reared in Los Angeles, Santa Ana, and Santa Paula from various introduced species of *Buddleia,* and Keifer presumed that the moth had moved into the area from a more tropical region. The species was believed to breed continuously throughout the year, larvae having been collected in April, July, and October.

In recent years the species has apparently become established around the San Francisco Bay area, our first record having been an infestation at San Bruno, San Mateo County in May 1949. During the last four years adults have been taken at lights in Berkeley, Alameda County, in October, December, February, and April (Powell, J. R. Powers, G. I. Stage) and in Pleasant Hill, Contra Costa County, in May (P. A. Opler).—A. E. PRITCHARD and J. A. POWELL, *University of California, Berkeley.*

[1] Keifer, H. H., 1936. Bull. So. Calif. Acad. Sci., 35(1):13.